third and fifth postoperative day, respectively. CAG on the 25th postoperative day showed recanalized grafts to LAD and OM, though grafts to PL and PD were still occluded (Fig. 2). Argatroban was transited to oral warfarin on the 13th postoperative day and she was discharged on the 51st postoperative day. Coronary computed tomography examined six months after surgery revealed no restenosis in PCI target lesions and grafts to LAD and OM were still patent. The patient has survived with no cardiac events for eight months since the operation and aspirin and clopidogrel have been continued.

3. Discussion

Perioperative graft failure in coronary artery bypass grafting (CABG) is a serious complication occasionally resulting in poor mortality and morbidity [1, 2]. Coronary thrombosis is one of causes of graft failure; especially HIT could sometimes cause a fatal clinical manifestation because of its rapid and serious progression. In adult cardiac surgery, the frequency of HIT is known as 1.0 – 2.4% [3]. The occlusion rate for saphenous vein grafts, but not arterial conduits, was significantly increased after CABG in HIT patients [4].

The diagnosis of HIT is based on its typical clinical picture. The ‘4Ts’ of HIT, i.e. the degree of Thrombocytopenia, the Timing of the platelet fall after heparin exposure, the presence of Thrombosis, and oTher causes for thrombocytopenia excluded, may be useful for assessing patients with suspected HIT [5]. In our case, the total points by the ‘4Ts’ assessment point system were six out of eight and HIT was highly suspected, though IABP and cardiac surgery were possible causes of thrombocytopenia and points did not counted in the category of oTher causes for thrombocytopenia. Heparin exposure in PCI might progress thrombocytopenia. Immediate therapy, i.e. cessation of all heparins including heparin-coated catheter and alternative anticoagulation with argatroban, was effective. Thielmann et al. reported that PCI was superior to redo-CABG for graft failure in limiting the extent of myocardial damage [1].

HIT-antibody, i.e. heparin-platelet factor 4 (PF4) antibody testing is recommended for confirming the clinical diagnosis of HIT; however, initiation of proper treatment must never be delayed pending laboratory results, which may not be obtained for hours to days. In our case, heparin-PF4 antibody measured on the second postoperative day was reported negative two weeks later. The assay in our institute was the ELISA and the sensitivity and specificity were limited to 73% and 77%, respectively.

HIT was considered most compatible for rapid and serious thrombocytopenia. Localized vascular injury and the direct contact to the foreign body, that is the intracoronary shunt tube used during anastomosis procedure, might play an important role for thromb formation in surgical site.

When HIT is diagnosed preoperatively, cardiac surgery should be delayed until heparin-PF4 antibodies turn into undetectable. Heparin is recommended over alternative anticoagulants because of the limited experience with the latter agents in cardiovascular surgery and their lack of specific antidotes and monitoring. If delay is impossible, cardiac surgery should be performed under alternative anticoagulants, though the safety and efficacy are not established. Antiplatelet therapy, such as glycoprotein IIb/IIIa blockade, combined with heparin or alternative anticoagulants has been reported and may be effective [3]. The experience is also limited and the risk of bleeding is a great concern. It should be applied with prudent consideration.

This is a clinically worthy case who was successfully treated by PCI and argatroban for inoperable acute coronary thrombosis in OPCAB.

References


EComment: Heparin-induced thrombocytopenia in cardiac surgery

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We appreciate your decision to transfer the patient to intensive care unit at the end of the intervention; a longer stay in the operating room carries major complications [1]. We agree also with your decision to perform percutaneous coronary intervention (PCI) and not redo coronary artery bypass grafting (CABG). Heparin-induced thrombocytopenia (HIT) can be developed in 1–2.4% of patients treated with heparin [2]. HIT is an antibody-mediated reaction caused by exposure to heparin and while it usually occurs 5–10 days from the heparin therapy it can happen more rapidly in patients who are re-exposed to heparin within 100 days [3].

We recently treated surgically a 78-year-old man with a history of coronary disease. During his hospitalization in the intensive care unit a severe thrombocytopenia was observed and HIT was diagnosed. Heparin-platelet factor 4 (PF4) antibody test confirmed HIT resulting positive (68%). We postponed CABG for 120 days but antibodies were still high (56%) at his re-admission. Antiplatelet antibodies were measured an ELISA immunoassay; test with sensitivity and specificity of 73% and 77%, respectively [1]. The management of patients with HIT who underwent cardiac surgery varies widely in the literature. Lepirudin, argatroban, bivalirudin are some of the agents used in case of HIT in cardiac surgery [1, 3, 4].

An OPCAB was performed. Fondaparinux sodium 2.5 mg once daily was administered during the two preoperative days and one single dose was given 30 min before anastomoses of the left internal mammary artery to the left anterior descending artery. The management of the particular patient was successful and the postoperative course uneventful.

Consequently, we suggest fondaparinux sodium as alternative treatment in cases where coronary disease and HIT co-exist, while more studies are needed for the stabilization of this suggestion.
References


